The University of Chicago

The Effect of Alcohol on the Intelligent Behavior of the White Rat and Its Progeny

A DISSERTATION

SUBMITTED TO THE FACULTY

OF THE

GRADUATE SCHOOL OF ARTS AND LITERATURE

IN CANDIDACY FOR THE DEGREE OF

DOCTOR OF PHILOSOPHY

IN THE

DEPARTMENT OF PSYCHOLOGY

By

ADA HART ARLITT

The Psychological Monographs
Vol. XXVI, Whole No. 115

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PRINCETON, N. J.
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THE EFFECT OF ALCOHOL ON THE INTELLIGENT BEHAVIOR OF THE WHITE RAT

Introduction

These experiments, begun October, 1914 and ending June, 1917, were undertaken with a view to determining (1) the effect of alcohol on the intelligent* behavior of animals to which it was administered; (2) the effect if any on the non-alcoholic offspring of these animals. The first of these problems was suggested by the apparent lack of social efficiency present in a large proportion of humans addicted to the use of quantities of alcohol in its various forms; the second by the number of mental and nervous anomalies present in the offspring of drinkers. According to investigators of the etiology of mental deficiency, temporary or permanent alcoholism on the part of one or both parents is frequently the cause of mentally deficient children. Of the cases examined by Beach and Shuttleworth† 16.38 per cent had parental alcoholism as the sole cause; of those examined by Tredgold‡ 46.5 per cent had parental alcoholism as either the sole or the chief cause. In the case of the human the question always arises as to whether alcoholism is the cause of neurotic instability or the expression of it. Our chief aim was to determine whether alcoholism alone, working on a thoroughly healthy stock, could produce abnormalities of behavior in the first generation and mental and nervous anomalies in succeeding generations.

In order to obtain a stock of known antecedents whose environment could be controlled it was necessary to use animals as our subjects.

It is not here maintained that results obtained from animals will enable us to draw conclusions directly applicable to humans,

* By intelligence is meant the capacity to learn—to utilize past experience in the process of adaptation to a new situation.

† Mentally Deficient Children, p. 80.

‡ Mental Deficiency.

but it is maintained that such results will throw some light on the problem.

White rats were chosen because they thrive and breed under laboratory conditions, because they will readily take alcohol with their food and lastly because, if the problem be suited to their organization, they learn with great rapidity. The maze problem has been used extensively to study the learning process in rats and it has been found excellently adapted to the sense organ and muscle capacities of these animals. Maze learning, therefore, was the problem chosen as a test of intelligence.

A cut of the maze used appears on this page. Each section retraced in the true path and entrances into blind alleys constituted errors. The maze was considered learned when four out of five runs had been made without error. The number of trials taken to master the maze and the total time taken and the errors made before this occurred were the criteria used in judging relative intelligence. The only motive for running the maze was hunger. Punishment was at no time used. In no case are the records of animals frightened at any time during the training period included in the group records.

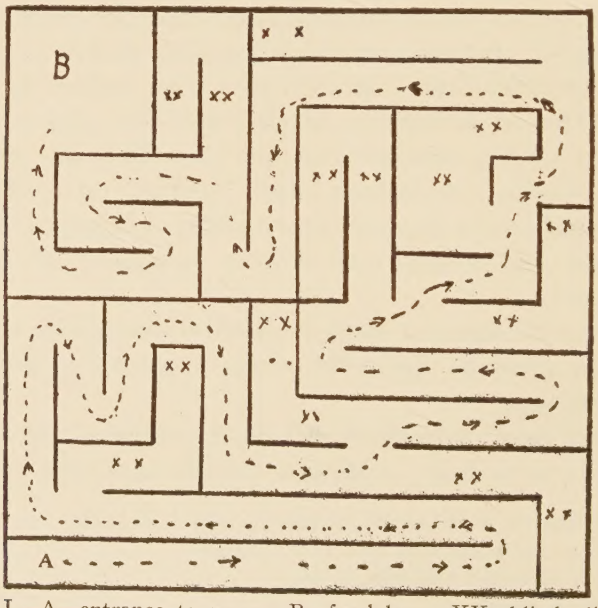

FIGURE I.—A—entrance to maze. B—food box. XX—blind alleys. Dotted line (.....) shows true path.

Rats receiving 2 c.c. and 2 ¼ c.c. for 3 months were left in the maze until they found their way out at each trial. As this took more time than it was possible to give later sets a different procedure was employed with all other animals including three sets which received the same amount of alcohol for the same length of time as the three groups trained by the first procedure.

The second procedure differed from the first in that rats which failed to find their way out in 30 minutes in all trials after the first were removed from the maze at the end of this time. Fifteen hours were allowed for the first trial, and also for the twenty-fifth provided the animal had not found its way to the food box in the 23 thirty minute trials between the 2 fifteen hour trials. All animals failing to learn under this treatment were termed failures and their records not included in the group average. It must be understood that no animal was removed from the maze that was trying to find its way to the food box. It was removed only after it had remained inactive for ten minutes or more. Certain animals found their way to the food box, then failed. These were given 24 thirty-minute trials and one 15 hour trial. If they failed to run the maze during these 25 trials, they too were termed failures and their records not included in the group average. As a matter of convenience the term "failure" will be used throughout the rest of this paper in referring to animals exhibiting this type of behavior.

Failures usually moved past two or three turns in the true path, then went into a blind alley. After making aimless movements for two or three minutes, they went to sleep and remained so until the end of the trial. A few did not move more than a foot from the entrance of the maze. Very few alcoholics and no normals failed. The usual procedure with animals was to leave them in the maze until they found their way out of their own accord.

Alcohol was administered to our subjects by the feeding method. It was mixed with the bread and milk fed once daily to all animals. This procedure differed from that used by the majority of previous experimenters but it was chosen rather than any other, because (1) the exact amount of alcohol consumed

by each group could be accurately measured; (2) it is possible to change the amount of alcohol administered without any radical change in the feeding procedure; (3) it makes the case of the rat more analogous to that of the human drinker.

Feeding methods heretofore used may be roughly grouped under four heads; stomach pump methods, in which the alcohol is introduced by means of the above-mentioned apparatus; fuming methods, in which eggs or mature animals are subjected to the action of alcohol fumes; absorption methods—in which eggs are placed in liquids which contain alcohol in solution; and feeding methods—in which the alcohol is given mixed with the food.

Hodge[4] used the stomach tube method with kittens, but found that respiratory tract diseases developed shortly after administration of alcohol was begun. This method is too difficult to use with small animals and is moreover as compared with feeding methods, unnatural and may induce emotional and other disturbances.

Variations of the fuming method have been used by a number of experimenters. Feré[2] experimented with the effect of fuming chick eggs on the percentage of chicks produced and the anatomical defects present. Chick eggs were subjected to the action of alcohol fumes, then incubated.

Stockard,[10] too, used this method with chick eggs to determine its effect on the structure of chicks hatched from eggs thus treated. It was used by Riddle and Basset[8] with pigeons to determine the effect of alcohol on the size of the yolk of the pigeon's egg; and by Pearl[7] with adult fowls to determine the effect of alcohol on the progeny of alcoholized fowl. Stockard[11] used the fuming method throughout his whole series of experiments concerning the effect of alcoholizing mammals on their progeny. In all of the above cited experiments the interest of the investigators was anatomical and in some instances physiological as well, but in no case purely psychological.

The method used in administering alcohol was so different from the feeding method as to warrant our expecting a different result from that obtained when alcohol was administered by the feeding method. Where eggs were fumed the embryos were in

contact with alcohol fumes in the early part of their development. This condition is not analogous to the effect on the embryo of alcohol in the circulatory system of pregnant females, nor to its effect on the reproductive system of males. The fuming method used with animals themselves does, of course, permit of the introduction of alcohol into the system of pregnant females, or males and females about to be mated, but by this method alcohol is introduced through the respiratory tract. This leaves something to be desired if results obtained from this method are to apply to the human drinker whose alcohol comes into the system by way of the digestive tract. It was for this reason more than any other that the feeding method was used with our subjects.

Though the technique and the animals used in the Stockard experiments differed from ours the author wishes here to acknowledge her debt to these experiments for many valuable suggestions.

The method of absorption was used by Stockard[9] with fish eggs, and by Fletcher, Abbott and Arlitt[3] with chick eggs. In the former case the eggs were placed in a solution of alcohol and sea water; in the latter alcohol was injected into the air space of the eggs, the opening made for the injection was sealed and the eggs were incubated. The interest in the first experiment was anatomical, in the second psychological. Though the interest in the experiments of Fletcher, Abbott and Arlitt was like ours, psychological, the animals used and the technique differ so widely as to render a discussion in this connection of little value.

Nice, working with the effect of alcohol on the growth, reproduction and daily activity of white mice, used the feeding method that most nearly approximates that used in our experiments. Three c.c. of 35 per cent alcohol was mixed every other day with the crackers and milk offered to his subjects and the animals drank 35 per cent alcohol instead of water. Our subjects drank only pure water and the amount of alcohol, and the length of the feeding period before testing the effect of the drug on the animals was varied from group to group.

In the experiments previously cited none are comparable to ours in both method and interest; while some used the feeding method and some studied the effect of alcohol on behavior, none did both things. To the author's knowledge there is but one experiment comparable to ours, that of Hodge.[4] Hodge fed whiskey, wine, beer, and diluted alcohol to puppies and adult dogs, then tested the intelligence of these animals as measured by ability to retrieve balls and to obey commands. He records also the reproductive capacity of the first generation and the viability of the second. His interest was anatomical as well as psychological but no attempt was made to test the reproductive capacity of any generation after the first. Our experiments differ from his chiefly in that the intelligence of the first, second, third, and fourth generations was tested as well as their viability, whereas Hodge records only the viability of the second generation and does not attempt to experiment with any further generations. Also, no attempt was made to vary the size of the dose, or the duration of feeding.

In only two of the experiments cited was behavior the object of the investigation and in none was the procedure used in administering the drug exactly the same as that used by us; ours is, so far as we know, the only experiment in which the problem was to determine the effect of various amounts of alcohol administered for periods of different lengths on the intelligent behavior of the first, second, third, and fourth generation of white rats.

Our subjects were all bought from the same animal dealer. They were male and female rats approximately 93 days old when alcohol feeding was begun.

In order to eliminate group or strain differences our animals were chosen, from 25 to 50 at a time, at random from large groups bought for use in the laboratory. The remaining rats in each group, numbering from 15 to 60, were divided between two experimenters on other problems. The rats chosen by us were again divided at random into smaller groups of from 7 to 18 each. One of these groups was used as a normal control, the others were fed ¼ c.c. to 3 c.c. of 95 per cent grain alcohol per

day per rat for from 16 days to 6 months before any tests of intelligence were made. This method of choosing groups eliminated the possibility that the differences in behavior manifest from group to group were due to strain differences. Our groups of normal controls chosen at random and all animals used by the two experimenters on other problems showed normal learning capacity. This could not have been the case in groups chosen at random, were any of the animals bought by us from defective strains.

All animals were kept 10 days before alcohol feeding was begun in order that any which had acquired diseases while in the care of the animal dealer might be eliminated. Only those in perfect health at the end of this time were used. One set was fed ¼ c.c., one ½ c.c., one 2 c.c., and one 3 c.c. of 90 per cent grain alcohol per day per rat for 16 days before tests of intelligence were made and throughout the testing period; four sets were fed ¼ c.c., ½ c.c., 2 c.c., and 3 c.c. respectively per rat per day for three months and two sets were fed ¼ c.c. and ½ c.c. for 6 months before tests of intelligence were made and during the entire testing period. Three normal control groups, raised under the same conditions, were run, one at the end of 16 days, one at the end of three months and one at the end of 6 months. These normal controls were the same age as the alcoholized animals. For the human of the same relative age and weight ¼ c.c. would be equivalent to 181.4 c.c., ½ c.c. to 362.8 c.c., 2 c.c. to 1451.2 c.c., and 2 ¼ c.c. (the dose actually taken by 3 c.c. rats) to 1632.6 c.c. of strong whiskey daily. The bread and milk to be given to each group was weighed and the alcohol to be administered added immediately before feeding. Twelve c.c. of bread and milk was allowed per animal except in the two cases where large doses were administered. Here 1 c.c. was deducted as the alcohol was considered to have the same effect as food. All food was given in china saucers and the animals fed on either a marble-topped, or a white oilcloth-covered table, to avoid absorption of the liquid content of the food. The maximum length of the feeding period was limited to 10 minutes in order to minimize, as far as possible, the evaporation of the alcohol.

Within the above stated limit, when rats left the food of their own accord, they were replaced in their cages. Food left by each set was weighed and a record made of the quantity. This amount was, except in the case of 3 c.c. animals, too minute to contain a measurable quantity of alcohol. Three c.c. animals actually averaged in amount consumed 2 ¼ c.c. of alcohol. These sets are therefore termed 2 ¼ c.c. rats. Animals were fed *after* each run in the maze. They were not under the immediate influence of alcohol while running.

I

Pathological Effects

Although our chief aim in this experiment was to determine the effect of alcohol on intelligent behavior, certain anatomical and physiological effects of the drug were so pronounced as to be worthy of note. Records of these were made primarily because of the light they might shed on the interpretation of our behavior data. They are given here, not only for the reason above cited, but because it was thought that they might be of value to readers whose interest was other than psychological.

Effect on Growth

An attempt was made to obtain records of the body weight and length of all animals used in the last year during which the experiment was in progress.

The apparatus used to obtain body lengths was a small box, one foot in length and two inches in width, with sides two inches in height. This box was open at one end and was without a cover. In the center of the bottom of the box was set a millimeter scale running lengthwise. The animal to be measured was placed in the apparatus with its nose touching the closed end and its body placed lightly against the scale. The body length from the tip of the nose to the root of the tail was then recorded. As the animals were very tame they could easily be held in the desired position. A difficulty presented itself almost immediately. The different states of muscular tension under which the animal was at the time it was measured caused a corresponding increase or decrease in the apparent length of the body. The records obtained proved so highly variable that they are not here included for discussion. The use of the caliper presented the same difficulty. This criterion, i.e. body length, was therefore not used in judging relative growth. Body weight was the only possible measure.

The animals were weighed on an ordinary apothecaries' scale. The weight was recorded in grams. Animals to be weighed were placed in a small, very light cage, the sides of which were made of wire and the bottom of wood. The cage was weighed before the rat was placed in it and its weight subtracted from the total weight of cage and animal. There were no difficulties to be surmounted in securing accurate weights, so it is on these that our conclusions concerning the effect of alcohol on growth are based.

As will be seen from the growth curves shown in Fig. II, rats receiving large doses, i.e. ¼ and 2 c.c. per rat per day, gained weight very slowly as compared with normals and in some cases actually lost weight though the animals were in the growing period. At the end of 9 weeks feeding, 2 ¼ c.c. rats weigh only 121.8 grams and 2 c.c. rats, though 10 grams heavier than normals when feeding was begun, only 130.1 grams, whereas normals at the end of the same period weigh 167 gms. At the end of 12 weeks the 2 ¼ c.c. group weighs only 116 gms. and the 2 c.c. group 125 gms., an average loss of 5 gms. per rat in the three weeks elapsing between the 9th and the 12th week. At this time normals weigh 188 gms., an average gain of 21 gms. per rat. Animals receiving ¼ c. c. and ½ c. c. per rat per day gain weight steadily, but show less gain at all stages than do normals.

Alcohol has its most pronounced effect on the growth rate of 2 c.c., 2 ¼ c.c. and ¼ c.c. rats at the end of the 12th week, that is to say, the most pronounced retardation occurs between the 9th and 12th week. It has an approximately equal effect on ½ c.c. rats from the beginning of the feeding period to the 9th week. In all cases alcohol causes a marked retardation in the rate of growth as judged by the disparity in body weight of alcoholics as compared with normals of the same age.

Effect of Parental Alcoholism on the Growth Rate of the 2nd, 3rd and 4th Generations

Parental alcoholism has a more marked effect on the rate of growth, than is present when the drug is administered to the animals themselves, as is obvious from analysis of the growth

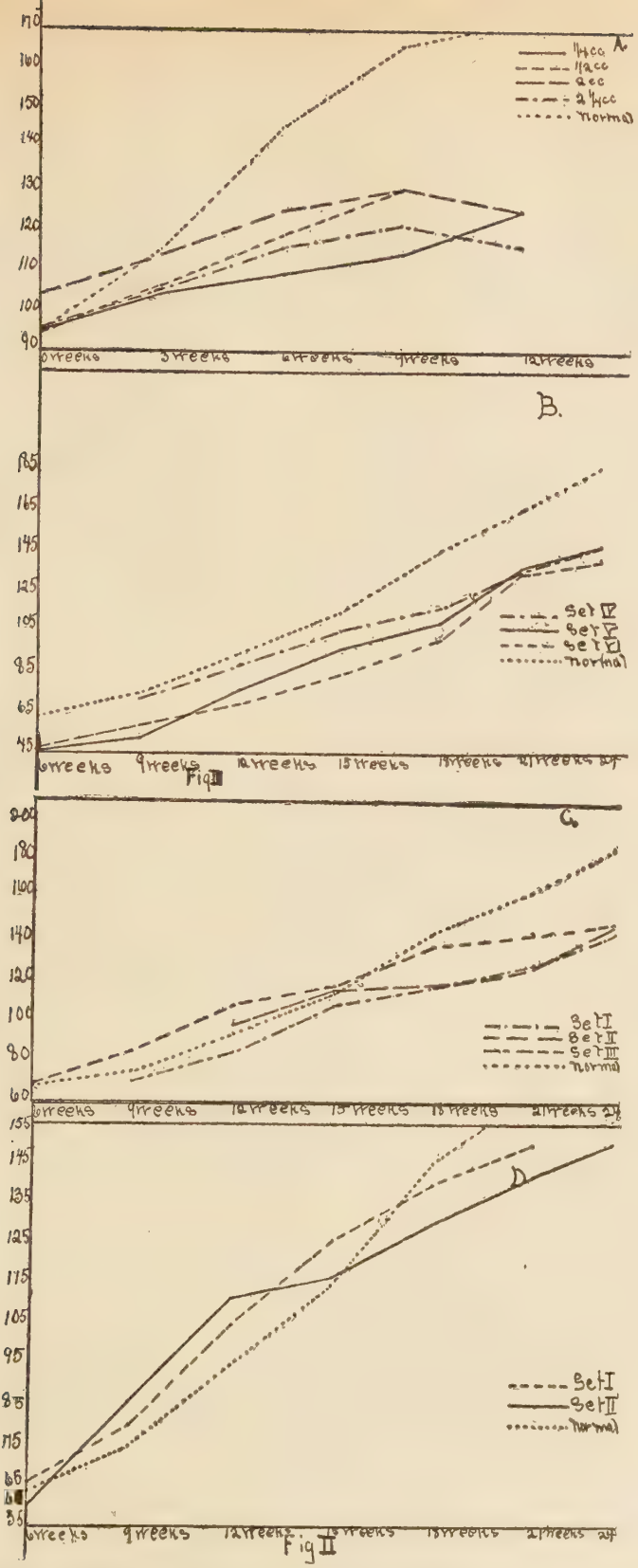

FIGURE II.—Effect of alcohol on the rate of growth. II A, first generation; II B, second generation. II C, third, and II D, fourth generation. [For amount of alcohol administered to each see text.]

curves shown in Fig. II. Retardation in the rate of growth is present to a marked degree in the 2nd generation and to a slightly less degree in the third and fourth. A comparison of the data contained in Figs. II B-IID (weights of 2nd, 3rd, and 4th generation rats) with the data contained in Fig. II A. brings out the following facts.

The defective body weight acquired by alcoholized rats is also inherited. The body weight of the second generation of alcoholic rats is below normal for the period of 6-24 weeks for which records were taken.

In the third and fourth generations the body weight approximates the normal until the 15th week and then drops below it. In all cases the body weight is below the normal from the 15th to the 24th week. Retardation of growth due to inherited defect is thus mainly evident at 15 weeks of age.

Comparing generations, it is evident from the graphs that the degree of retardation up to the 15th week becomes less and less with succeeding generations until the normal weight is attained. In other words the defect is eliminated by breeding for this period of age. This factor, elimination by breeding, will be discussed more fully in another section but it may be well here to state that we have anatomical evidence that this elimination occurred, if by 'elimination by breeding' one means that the germ cells themselves were in many cases so defective that young could not be produced from them; so that young of the second generation represent only those germ cells which were able to survive treatment with alcohol, or presumably the stronger cells. This elimination occurred for all generations succeeding the first. Only a few animals were sufficiently resistent to the drug to reproduce and of these only a few had offspring all of which survived.

For the period of 15-24 weeks the evidence is not conclusive, but as it is it indicates that the deficiency decreases from one generation to the next.

As to the relation of inherited defect to the amount and duration of the dosage the evidence is not absolutely conclusive, but in the majority of the cases the greater dosage is associated with

the greater defect. In the two cases of hybrids (the offspring of matings between alcoholics and normal animals) both are below the normal curve of growth. As compared with abnormals, one is above, the other below the growth curve of animals both of whose parents were alcoholic.

Effect on Longevity

Data on the longevity of animals to whom alcohol was administered are given in Table I. Records were not kept for a period greater than 6 months, for at the end of the training period, the surviving rats were subjected to an autopsy.

Alcohol exerts a pronounced effect upon length of life. From 10 per cent to 82 per cent of the alcoholized rats died within six months, while no deaths occurred within this period in the control sets of normal animals. The number of deaths due to alcohol varies directly with the dosage. The percentages are 10, 24, 74, and 82 for the ¼ c.c., ½ c.c., 2 c.c., and 2 ¼ c.c. dosages respectively. It was found impossible to test the intelligence after 6 months feeding of the two larger dosages, as only one of the animals subjected to these two dosages survived for this period of time. This animal lived for 7 months after feeding began.

Alcohol exerts its effect very quickly. The number of deaths during the first week of feeding is approximately as great as that for any subsequent period. For the ½ c.c. and ¼ c.c. the number of deaths for the first and last halves are practically the same. The absolute number of deaths for the two larger doses is greater for the first half of the feeding period, but in proportion to the number in the group the mortality for the first half is slightly less than that for the second. However, the number of deaths is far from proportional to the length of the feeding period.

No records were kept of the longevity of the 2nd, 3rd, and 4th generations as all of these animals were subjected to an autopsy as soon as they had mastered the maze.

Effect on Fertility

Male and female rats of all alcoholic sets were mated with animals of the same set and with normals. The procedure fol-

TABLE I

Effect of Alcohol Feeding on Length of Life

Duration of Feeding Before Death	Normal Rats	Total No.	Deaths ¼ c.c.	Total No.	Deaths ½ c.c.	Total No.	Deaths 2 c.c.	Total No.	Deaths 2 ½ c.c.	Total No.
(Weeks)										
1	0		1		0		5		3	
2	0		1		1		8		13	
3	0		0		0		8		5	
4	0		0		2		10		5	
5	0		0		1		4		7	
6	0		0		0		0		9	
7	0		0		0		3		1	
8	0		0		0		0		5	
9	0		0		0		3		4	
10	0		0		0		4		2	
11	0		0		0		8		4	
12	0		0		0		2		4	
13	0		0		0		5		4	
14	0		0		0		1		4	
15	0		0		0		No sets of 2 c.c. and			
16	0		0		1		2 ¼ c.c. fed 6 mos.			
17 to 26	0		0		0					
22	0		2		4					
24	0		0		0					
Total	0		4	37	9	37	61	82	68	82
Percentage of Deaths		0%		10%		24%		74%		82%

lowed with the first three alcoholic sets was to mate at that point in the feeding period at which intelligence was to be tested. As no young resulted when mating was restricted to a specific time, males and females of alcoholic sets were left in the same cages throughout the feeding period except where matings with normals were desired. Abnormal females which were to be mated with normal males and normal females which were to be mated with abnormal males were kept in cages separate from males of their set for a period of three weeks before mating.

The 2 ¼ c.c. males and females were without exception sterile, both when mated with rats of the same set and when mated with normals. Six 2 ¼ c.c. males and a like number of females were left in the same cage for from one to three months (a pair to a cage) and during this time from one to three matings occurred. Two abnormal males mated with two normal females and two abnormal females mated with two normal males produced no young though left in the same cage for three months.

A like procedure was followed with 2 c.c. animals. Three 2 c.c. males were left in the same cage with three females of the same set for three months. During this time four matings occurred, but no young were produced. The same procedure was used with two abnormal males and two normal females and with two normal males and two abnormal females, but these also were without offspring. The only young born to 2 c.c. males were those produced after alcoholization had ceased for two months and the males were mated with normals.

Two 2 c.c. males mated with normal females after a feeding period of 5 months and a succeeding non-alcoholic period of 2 months produced litters of six each. One litter was eaten soon after birth. Two of the 6 young of the second litter were born dead. The four remaining young lived to be run in the maze. Too few males survived to render a repetition of this variation in feeding procedure advisable. Both females had normal litters before and after the mating with abnormals. The 2 c.c. males had no other offspring though mated twice during the alcoholic period and twice during the non-alcoholic.

As it was impossible to obtain young from 2 c.c. and 2 ¼ c.c. rats fed for even 10 days before mating, 4 normal females were mated with normal males. Ten days later 3 of these were fed 2 ¼ c.c. and 1, 2 c.c. per day per rat. Two of the first three had abortions one week later, one had a litter of 6 at full time. Of these 2 were born dead, 4 died when 13 days old. The 2 c.c. female had 5 young. One was abnormally small, one was still born, 2 died when two weeks old, and one when one month old.

As a variation of this experiment 2 normal males, never before alcoholic, were fed 2 c.c. and 2 were fed 2 ¼ c.c. and each mated with one normal female four hours later. Three litters resulted. One 2 ¼ male had no young though mating occurred. The litter of the second 2 ¼ c.c. male consisted of 5 young. One was still born, two died when 3 days old, and one died when three weeks old. The single surviving animal, a male, was run in the maze but completely failed to learn it. The 2 litters of 2 c.c. males consisted of four and six respectively when found by the experimenter approximately 18 hours later. Two of the

animals in the litter of four died when 6 days old, one died when 2 weeks old, the last when 5 weeks old. The litter of 6 lived only 2 days.

Though no conclusions could be based on the results of these variations alone, they serve to throw more light on the effect of alcohol on fertility and on the viability of the young produced.

It was difficult to obtain young even from animals, receiving so small a dose as ½ c.c. per day per rat. Only two, and these still-born, resulted from attempts to inbreed the first 6 animals fed that amount for 6 months. Four matings between ½ c.c. males and females fed 6 months produced no young and no young were born after 9 months of alcoholization.

Of the second ½ c.c. set 3 females had litters by males of the same set. One had 3 young at the end of 5 months feeding. Two of this litter were born dead; one died 2 weeks later. One had 6 young after 5 ½ months feeding, and one after 6 months. Two in each litter were born dead and the 4 remaining in the second were eaten 2 weeks after birth. A fourth female had a litter of 6 young by a normal male after 6 months feeding. Two of these were still-born. The 4 remaining young did not live to be trained in the maze. One ½ c.c. female mated with a normal male after a feeding period of 4 months produced 7 young. One was still born, the remaining 6 lived to be trained in the maze.

The ¼ c.c. animals had more litters than rats receiving larger doses, but only a few of these survived. One female had 5 young after 6 ½ weeks feeding; one had 7 young after 3 months by males of the same set. In each case all died the following day. One female had 7 young by a male of the same set after 6 months feeding. Four died immediately after birth; three lived to maturity. This same rat mated to a normal male 7 weeks later had 6 young, all of which died the following day. The weather was unusually severe and the laboratory very cold. This is the only case of death of second generation which can be directly ascribed to external conditions. One set of 4 young, born of parents fed ¼ c.c. for 39 days, one set of 9 born of 2 normal females and a ¼ c.c. 6 months male, and one set of 5

young born of ¼ c.c. four months parents, together with the first set discussed, numbering 3, born of ¼ c.c. 6 months parents, were the only surviving offspring born of the matings between ¼ c.c. rats and those of the same set and 4 cross matings between these animals and normals.

This tendency to sterility in alcoholized animals stands out all the more clearly when contrasted with the fertility of normals of the same age. In the period covered by the above report 2 sets of normal animals of 6 each had 38 healthy young born of 5 matings, exclusive of those born of cross matings with alcoholics. Only three of these young died. In addition to the 38 healthy young, two normal females had litters, one of four, the other of 5 young, which were eaten immediately after birth. Fifteen of the surviving young were trained in the maze at the same time as the abnormal second generation animals; the remaining 20 were given to two experimenters on other problems.

Set I of the second generation (male parent 2 c.c. 5 months, no alcohol 2 months; female parent normal) was sterile though mated twice. Set II of ½ c.c. 5 ½ months parents produced 13 young when inbred, nine from the first mating and 4 from the second. No young resulted from crossing the males of this set with normal females, as the males died soon after the first mating. Eighteen young were born of a mating between a normal male and the two females.

Sets III and IV whose parents received ¼ c.c. for 39 days and 4 months had 12 and 13 young, respectively, when inbred. Eleven of each have been trained in the maze. One male of Set III and one of Set IV had litters by normal females of 4 and 6 each respectively. One of the former litter died soon after birth, all of the latter survived. Set V was also sterile though twice crossed with normal males and twice with normal females. As Sets VI and VII were born towards the close of the experiment, no attempts were made to test them for fertility.

Set II of the third generation, parents Set III of the 2nd generation, produced one litter of 7 apparently normal young. No further tests of fertility were made as these animals were killed for pathological examination.

There is apparently a selective process at work in the case of Set III third generation, the offspring of Set II second generation. Only one male and one female had young, though attempts were made to inbreed and to cross other males and females with normals. Three males and 3 females of Set I were left in the same cage together for 6 months. Two males were left in the same cage with normal females and two females with normal males for three months. The author observed no mating in any of these cases and no young were produced though the animals were left together at the mating season. With the exception of this set of the third generation and Set I of the second generation no tendency to sterility was present in the surviving offspring of alcoholized animals. The surviving 2nd generation animals were, as will be seen when the results of the pathological material which follow have been studied, probably a highly selected group.

To sum up the results of alcoholism on reproductive capacity:

(1) Alcohol produced complete or partial sterility in those rats to whom it was administered.

(2) The degree of sterility is proportional to the size of the dose. Our results allow no confident assertion as to the relation of the degree of fertility to the duration of feeding.

(3) The sterility effected characterizes both males and females, since both are sterile when mated with normal animals.

(4) This sterility is not due to lack of sexual desire as a normal number of matings occurred.

(5) This sterility is not a seasonal affair as normals of the same age bred successfully at the same time.

(6) Alcohol produces abortions in pregnant females.

(7) The degree of sterility is partly overcome by stopping the feeding.

(8) When conception does occur, alcohol increases the number of still births and increases infant mortality.

(9) This sterility and lack of viability of the offspring is inherited to some degree by the second and third generation. The degree of inherited defect is proportional to the degree of acquired sterility.

(10) With ½ c.c. and 2 c.c. animals the inherited defect is greater than the acquired sterility and the degree of sterility keeps increasing with successive generations. There is no recovery from the induced sterility, i.e. it does not breed out.

(11) The defects due to alcohol are eliminated by the non-production or elimination of offspring, that is, what occurs is the elimination of the tainted stock rather than an elimination of the taint while the stock remains.

The Effect of Alcohol on the Internal Organs

It was not until an examination of the internal organs of alcoholized rats was undertaken by Dr. Wells of the Department of Pathology of the University of Chicago, that the cause of the difference in fertility between alcoholized and normal animals was made clear. An examination was made of the testicles, stomach, heart, lungs, kidney and adrenals, and the liver of male animals fed from ¼ c.c. to 2 ¼ c.c. per day per rat, as well as their offspring. A comparison of the results obtained from these examinations with those obtained from the examinations of the same organs of non-alcoholic animals brought out the fact that only the generative tract of alcoholics showed definite changes, but here the effects were so marked and so nearly constant as to stand out conspicuously as resulting from alcoholization. The seminiferous tubules of alcoholic rats were on the average much smaller than those of normals. Not only was this the case, but equally definite changes were apparent within the tubules. These results have been published separately and we merely state the general results in this paper. To quote from the article, "The changes produced by alcohol take place in quite definite order. At first the spermatocytes seem to be normal in number and appearance, but there is soon noticed an increase in the number of spermatids in the tubules with a decrease in the number of spermatocytes. At the same time, or earlier, there is observed a greater diminution of complete spermatozoa with tails, than in the number of sperm heads.

"Apparently the first effect of the alcohol is to render the formation of spermatozoa incomplete so that heads are formed without normal tails. The next effect of the alcohol seems to be to prevent the transformation of the spermatids into spermatozoa, whereby the tubules become filled with accumulated spermatids with but few spermatozoa or none at all. In the most

advanced stage the tubules contain but marginal cells with but few or no spermatocytes or spermatids." In this paper it is noted that the defects are the same as those present in the human alcoholic.

Before complete sterility occurs therefore, the animal is producing spermatozoa which show all degrees of abnormality. The connection between this condition of the male generative organ and total sterility or the production of defective offspring is obvious. Not all rats were affected alike which accounts for the absolute sterility of some animals as compared with the ability to reproduce present in others fed the same amount of alcohol for the same length of time.

The exact condition of the female generative organ, the ovary, of alcoholized animals is not known, though a brief examination of the same number of ovaries as testicles, i.e. 15, was made. Of the 15 ovaries, 2 were atrophic and several showed less ova than normal, but only an examination of serial sections which the authors were unable to undertake, would have revealed the exact changes present in alcoholized females as contrasted with normals. It is only fair to assume in view of the tendency to sterility present in females receiving large doses and the number of still births among the few litters produced by females fed small doses, that some changes other than those already noted were present.

II

The Effect of Alcohol on Habits Acquired Previous to its Use

Our first problem in studying the effect of alcohol on intelligence was to determine the effect of the drug on habits acquired previous to its use.

Three sets of animals were given two trials a day in the maze until all the animals in each set were making 8 out of 10 runs without error. The maximum number of trials necessary to attain this standard was found to be fifty. At the end of 50 trials, Set I was fed 2 c.c. per rat and Set. II 2 ¼ c.c. per rat every day and run in the maze daily as usual. Set III was used as a normal control. The alcohol was administered after the runs in the maze had been made.

The graphs in Fig. III show the results obtained from these groups.

Alcohol exerted no appreciable effect on the number of errors made. For this reason no graph or table of errors are given. At times the error records of alcoholic rats are above the normal about .3 of an error per trial, a result that may well be due to group differences. Alcohol thus decreases the accuracy or perfection of a well automatized act very slightly if at all. The time records for groups as a whole are given, Fig. IIIC, together with graphs showing the relative effect of alcohol on animals which have a high degree of resistance to the drug and on animals which succumb shortly after alcoholization begins (Fig. III A and B). It was thought best to plot the curves in terms of the average time for 10 trials rather than to show the irregularities which are present even in the time curves of normals when these curves represent variation from day to day.

As compared with normals, both groups of alcoholic rats show a marked decrease in speed after alcoholization begins, but the initial decrease does not occur for both groups at the same time,

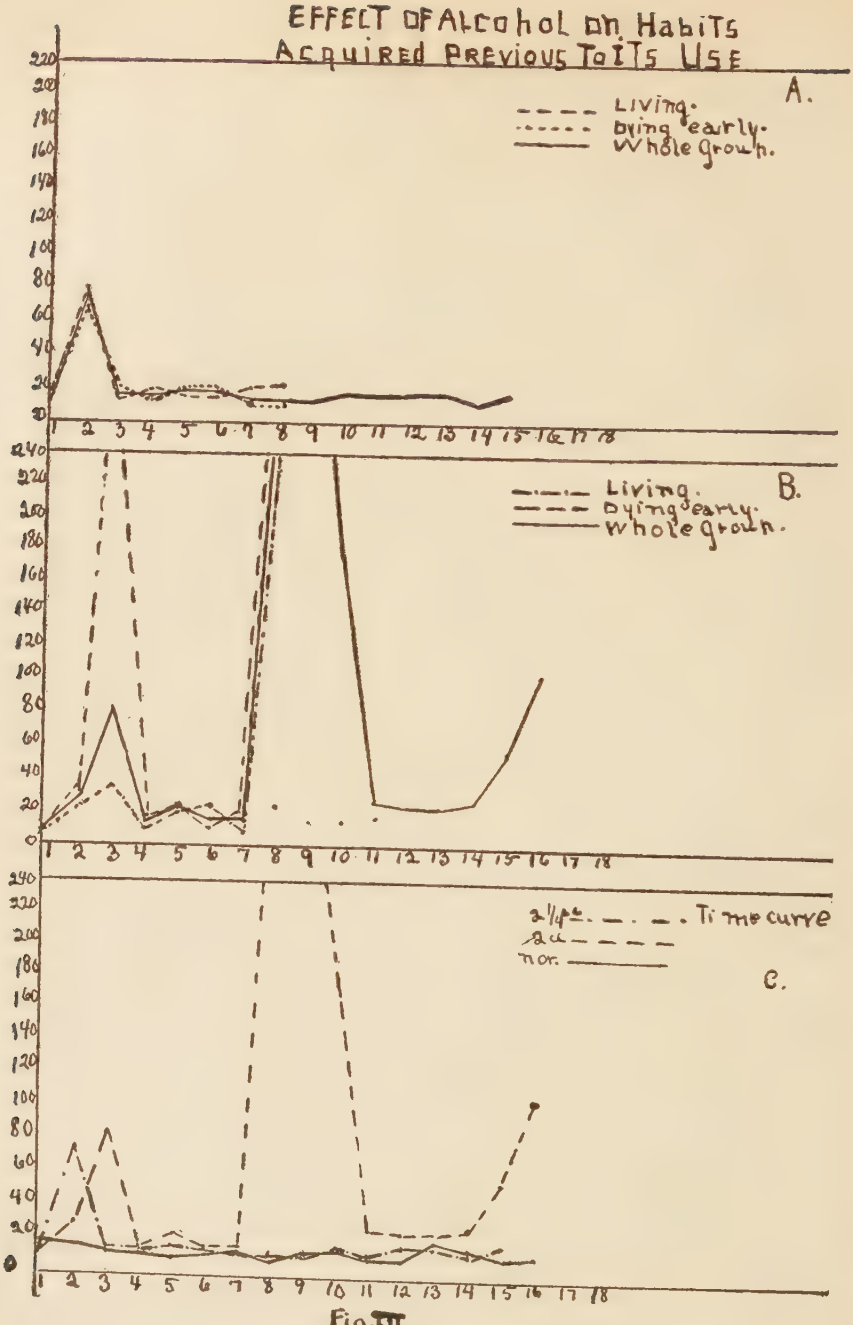

FIGURE III.—A—The effect of 2¼ c.c. per day, per rat, on the functioning of a habit formed previous to its use, in rats surviving and in those dying shortly after alcohol administration signs. B—Same where the dose is 2 c.c. per day per rat. C—comparison of a normal curve with curves showing the effect of 2 ¼ c.c. and 2 c.c. per day per rat on the groups as a whole. Unit for time curve, one second.

nor is the behavior of the two sets subsequent to this the same. The 2 ¼ c.c. set showed the effect of alcohol at once. These rats failed frequently in the 10 trials after the first feeding: that is to say, they did not move more than one foot from the entrance to the maze for two 15 minute trials per day. This tendency to inhibit movement was the most noticeable effect of the alcohol. At the end of 20 trials some accommodation was present and from that time until the end of the feeding period these animals were no slower than a slow normal. The 2 c.c. rats did not show a marked breaking down in the maze habit until approximately two weeks after feeding had begun, at which time two rats died. They then showed accommodation to the effects of the drug for the next 40 trials, approximately three weeks. At the end of 6 weeks there is again accommodation which disappears for 20 trials before death occurs.

The experimenter was forced to be away from the laboratory 15 days. During this time the rats were in charge of an assistant.[1] Set I was run by this assistant from the 13th to the 15th trial[2] inclusive; Set II was run by her from the 5th to the 7th trial, and Set III from the 9th to the 11th trials inclusive. There was apparently no change in behavior due to the introduction of a new experimenter. This is probably due to the fact that the animals were too familiar with the maze situation to be easily disturbed by changes in external factors.

From the facts already cited we may safely conclude that alcohol does lessen the speed of performance of a habit acquired previous to its use. This lessened speed is not the result of working with a selected group. Except for the first rise in the time curve of 2 c.c. rats (Fig. III B), directly traceable to the slow running of two rats both of which died, there is no evidence to show that animals on which the drug has a slowing effect die before less affected animals. The curves for 2 ¼ c.c. rats of low and high resistance are practically synchronous (Fig. III A). Especially is this tendency of the two curves to overlap noticeable,

[1] Miss Moyer, a graduate student in the Department of Psychology, consented to run the animals in the maze during the experimenter's illness.

[2] By trial is meant, in this connection, point on the curve.

when it is remembered that they are not plotted in terms of large units, but in terms of seconds. Evidently the initial decrease in speed is due to the effect of the alcohol and the subsequent return to normal on the part of 2 ¼ c.c. rats and the partial return to normal on the part of 2 c. c. animals to accommodation, rather than to an elimination of animals in which the drug causes the most diminution in speed.

The results contained in this section show that alcohol does interfere with the functioning of habits acquired previous to its use in the direction of speed of performance. It does not interfere with accuracy to any appreciable extent.

There is a difference in the effect of the two doses. The animals showed a greater tendency to accommodate to 2 ¼ c. c. than to 2 c.c. doses. The previously cited differential effects are not due to the elimination of animals on which alcohol has the most pronounced effect in the direction of lessened speed, except in the case of the initial rise in the 2 c.c. curve (Fig. III C.).

Introduction of a new experimenter had no apparent effect on the behavior of alcoholized animals.

III

The Effect of Alcohol on the Intelligent Behavior of the First Generation

As compared with normals, alcoholics show decreased learning capacity according to all three criteria, viz., trials, total time, and total errors in 29 out of 30 cases (see Table II). The one exception refers to the errors for ¼ c.c. 10 days. With this single exception alcoholized animals took more trials and a longer time to master the maze and made more errors than normals of the same age raised under the same conditions.

Alcohol exerts its most pronounced effect on total time. At the end of 16 days feeding ¼ c.c. rats took 3018.3″, ½ c.c. rats took 9598.8″, 2 c.c. rats took 4316.6″, and 2 ¼ c.c. rats took 9767.5″ to master the maze as compared with the 1099.5″ taken by normals. At the end of three months feeding the disparity between the time required by normals and that required by alcoholics is even greater. Normals take 1131.6″, ¼ c.c., ½ c.c., 2 c.c., and 2 ¼ c.c. rats take 12016.2″, 2895.8″, 85406.2″, and 13022.2″ respectively. The total time taken by ¼ c.c. and ½ c.c. animals fed 6 months is 26481.6″ and 2397 respectively—as compared with the normal record of 1763″. This increase occurs not only for total time but for average time per trial as well, and the increase is not due to alcoholics taking a longer path. In 7 out of 10 cases the average error per trial made by alcoholics is less than that made by normals. In these cases the alcoholics actually took a shorter path. The increase in time over normals is due to the fact that alcoholics move very slowly and sometimes stop altogether. The most marked effect of the drug is therefore to lessen speed. The fact that alcoholics make fewer errors per trial than normals indicates that there is a diminution of exploratory tendencies.

The effect of the drug on trials and total errors is approximately equal. Both show, as has already been stated, a marked

TABLE II

Effect of Alcohol on the Intelligent Behavior of the First Generation

No. failing	No. learning	Amt. of alcohol	Total trials	M.V.	Total errors	M.V.	Total time	M.V.	Ave. time per trial	Ave. error per trial
					16 Day Group					
0	16	¼ c.c.	11.3	5.7	52.3	32.8	3018.3	3828.2	267.+″	4.6
0	17	½ c.c.	15.4	5.1	84.8	38.8	9598.8	10450.5	619.4″	5.5
4	9	2 c.c.	10.6	5.+	79.0	32.0	4316.6	5753.4	407.0″	7.4
2	6	2 ¼ c.c.	10.5	3.5	100.6	41.2	9767.5	9030.0	930.+″	9.5
0	5	Normal	6.7	1.3	55.5	17.0	1099.5	675.0	164.0″	8.2
					3 Mos. Group					
1	10	¼ c.c.	13.9	9.1	79.8	38.0	12016.2″	15748.9″	863.8″	5.7
0	5	½ c.c.	16.0	6.6	93.6	37.7	2895.8″	2816.0″	180.9″	5.8
2	6	2 c.c.	16.1	5.2	82.3	43.0	85406.2″	11552.5″	5304.1″	5.1
1	5	2 ¼ c.c.	20.8	5.3	120.2	24.7	13022.2″	11471.5″	626.0″	5.7
0	6	Normal	10.6	3.5	63.5	21.0	1131.6″	435.7″	106.7″	5.8
					6 Mos. Group					
1	5	¼ c.c.	20	11.0	133.2	61.4	26481.6″	894.2″	1324.0″	6.6
2	4	½ c.c.	14	7.0	70.5	22.0	2397.5″	854.2″	1498.0″	4.4
0	6	Normal	10.5	3.0	57.6	14.8	1763.3″	899.1″	167.+″	5.4
					3 Mos. Group					
					*Trained with First Procedure**					
0	6	½ c.c.	20.1	8.2	269.8	186.6	13822.6″	8320.1″	658.2″	13.4
0	5	2 c.c.	16.8	11.6	101.4	48.8	3260.0″	3412.8″	194.4″	6.0
0	6	3 c.c.	19.1	11.5	289.2	175.0	35298.0″	30694.7″	1844.4″	15.1

* As this procedure was used only with this group no discussion of results obtained therefrom is attempted.

increase over the normal indicating poor capacity to eliminate errors.

Lastly, alcohol increased group-variability. There was a marked difference in individual susceptibility to the drug even in sets fed the same amount of alcohol for the same length of time. The individual range of ability as expressed by the m.v. is, in 28 out of 30 cases, far greater than that in normal groups. The exceptions are for the times of the 6 months sets. The increase in variability would be greater and the exceptions eliminated were the records of the failures included. Failures are the extremes of group variability. The behavior of these animals has been described in a previous section. They could not learn the maze even when given two 15 hour and 24 thirty minute trials. Of the 13.2 c.c. rats fed 16 days, four, or 22 per cent, failed; of the eight 2 ¼ c.c. rats, 2 or 25 per cent, failed. Of the eight 2 c.c. rats fed 3 months before training, 2 or 25 per cent failed; of the six 2 ¼ c.c. rats, one or 16 per cent failed. At the end

of 6 months feeding, 16.6 per cent of ¼ c.c. and 33 ⅓ per cent of the ½ c.c. rats failed. There had also been one failure in the group of ¼ c.c. animals fed 3 months. There is no correlation between the length of the feeding period and the number of failures per group. As to the relation existing between the number of failures and the size of the dose, the two larger doses produced the greater number of failures.

The greater range of variability in alcoholic groups may be due to the larger number of animals in these sets as compared with normals. That is, variability may be merely a product of relative size. Against this possibility we would urge two points: the records of sets as set forth in the tables are the result of combinations of smaller sets of from three to eight in number and where the sets contain as many as five animals and in one case where the set numbered only 3, the variability was still far larger than in normal sets; second, with the two exceptions of the times for 6 months sets, the records as contained in the table for sets numbering the same or even less than the normal sets show a wider group variability. We believe, therefore, that what we have is a characteristic effect of alcohol on group variability due to differences in individual susceptibility to the drug.

Two of the previously noted effects of alcohol, decreased speed and increases in trials and total errors, are roughly in proportion to the amount of the drug consumed.

It might have been expected that where the duration of feeding was the same, the effect of the drug would be in proportion to the amount administered. In the 16 day group this does occur. The ¼ c.c. rats are better than ½ c.c., ½ c.c. than 2 c.c., and 2 c.c. than 2 ¼ c.c. The time, trials, and errors of the 2 c.c. set would show marked increase were the records of failures included. Adding the records of the four failures to the group average of 2 c.c. animals and the two in the 2 ¼ c. c. group to that average, produces a record which shows a gradual decrease in learning capacity directly in proportion to the amount of alcohol per rat. (See Fig. IV.)

The number of failures in the 2 c.c. group as compared with the 2 ¼ c.c. rats can be better explained in terms of results ob-

tained from the animals used in Section II. The drug, as has been previously stated, did not affect the behavior of 2 c.c. rats until two weeks after the first dose. At this time a number failed. The 16 day animals in Section II were probably feeling the worst effects of the drug at the beginning of the training

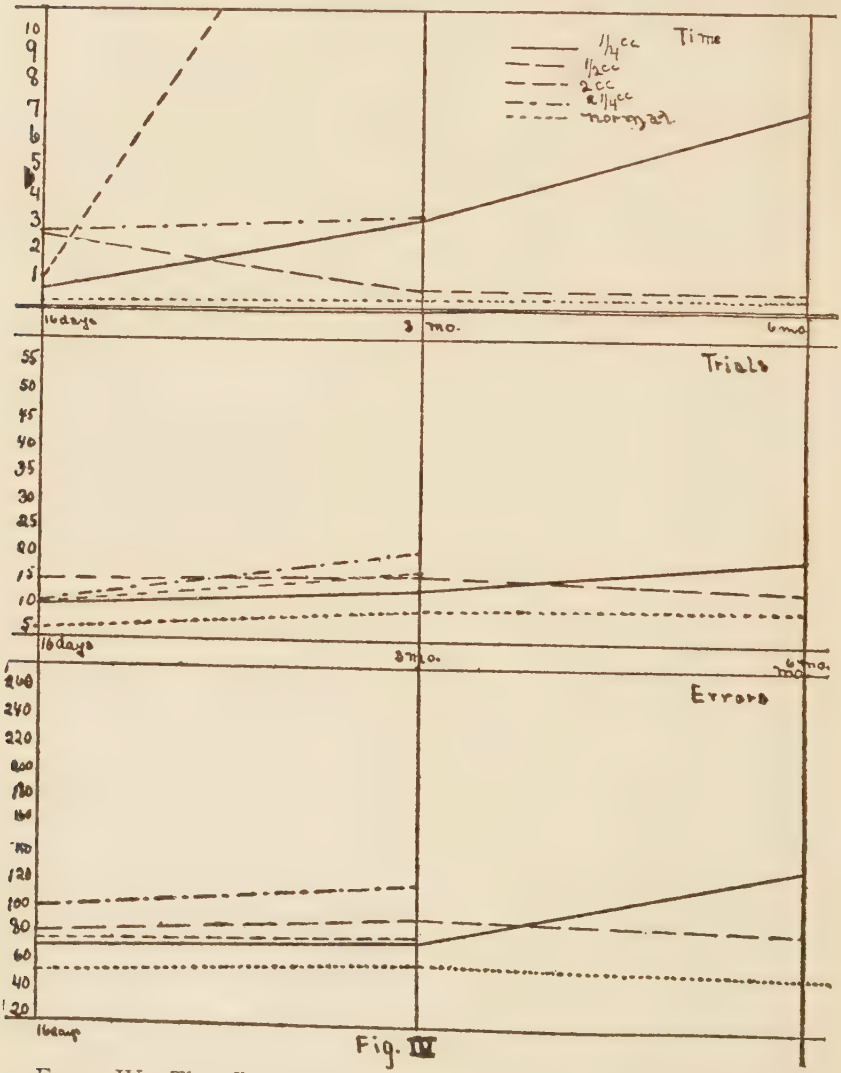

FIGURE IV.—The effect of duration of feeding with large, small, and medium doses of alcohol on time, trials and errors. Unit for time curve, one hour; for errors, ten; for trials, one.

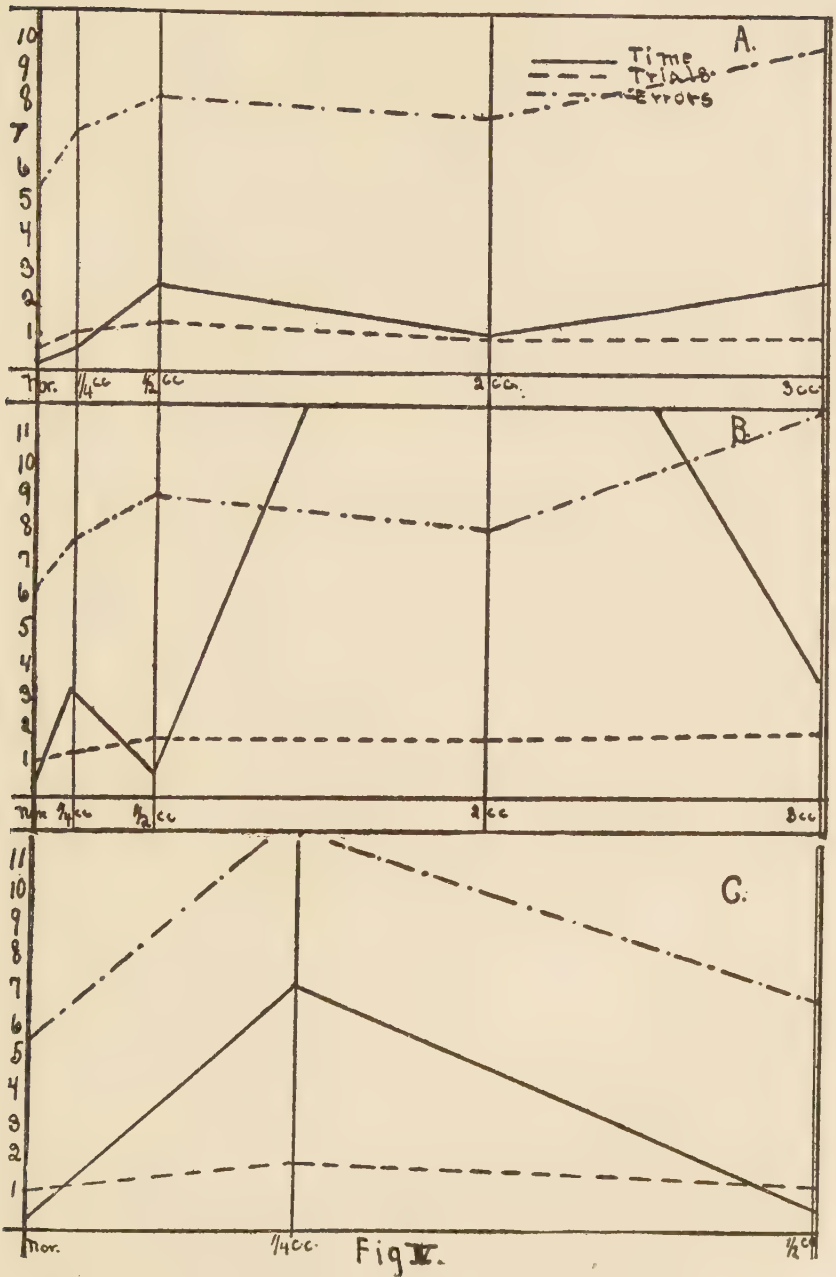

FIGURE V.—The relative effect of large, small, and medium doses of alcohol on time, trials, and errors, where the feeding period is of the same length. Unit for time curve, one hour; for errors, ten; for trials, one.

period and at that time acquired the habit of failing instead of that of learning the maze.

The 3 c.c. group in Section II showed the worst effects of alcohol the day after the first dose, then rapidly accommodated to it. If a like condition existed in the 2 c.c. and 3 c.c. animals used in Section III, the probability of failures in the case of the former would be much higher than in that of the latter. This seems a plausible explanation of the only peculiarity present in the records of the 16 day rats.

At the end of three months feeding many peculiarities appear in the records. Comparing the alcoholic sets, set for set with animals receiving the same amount for 16 days, all 3 months sets with one exception show an increase in all three measures over animals fed the same amount for 16 days. The one exception refers to the time for the ½ c.c. three months set (Fig. IV). Comparing alcoholic sets fed 3 months with each other (Fig. IV-V) and with normals, ½ c.c. rats take less time to master the maze than do ¼ c.c. animals, whereas 2 c.c. rats take longer to master the maze than do 2 ¼ c.c. animals. All are slower than normals. That is to say, if the animals were ranged in order as to speed of acquisition, ½ c.c. animals would come first after normals, ¼ c.c. second, 2¼ c.c. third, and 2 c.c. animals last, and adding the records of failures to the group average would only increase the disparity between ¼ c.c. and ½ c.c., and between 2 c.c. and 2 ¼ c.c. animals. The unusual speed of acquisition of ½ c.c. rats as a group was due to the records of two animals on whom alcohol seemed to have the effect of a stimulant to activity instead of its usual effect, i.e., a decrease in activity. The long time taken by 2 c.c. rats has as its explanation the strong tendency to fail manifest in all 2 c.c. sets. Of the 6 learning rats, 3 failed for from 2 to 15 trials, then learned the maze. Failing rats, as has been previously stated, average only 3 or 4 errors per 30′ trial. They do, however, have some opportunity to adapt to the maze situation even with this minimum of movement. The strangeness of the situation wears off and any fear which may have been present during the first two or three trials, tends to disappear. The records as to number of

trials taken to learn the maze and as to errors made during and after failing trials would tend to show slight difference, if any, over those of rats at no time failing, thus making the record of the group as a whole poor as to time and relatively good as to trials and errors.

In the other two measures, trials and errors, with the records of failures included there is a gradual decrease in learning capacity in proportion to the amount of alcohol administered. In all three measures alcoholics are markedly inferior to normals. (See Fig. IV.)

There are only two alcoholic sets in the 6 months group, a ¼ c.c. set and a ½ c.c. set. As compared with normals these animals are much slower and make many more errors, but the disparity between ½ c.c. animals and normals is not so great after 6 months as it was after 3 months feeding. Only the ¼ c.c. set has shown progressive deterioration. In all three measures, time, trials, and errors, the set fed ½ c.c. 6 months is better than the set fed ½ c.c. 3 months. (Fig. V.) Either it is better to take a moderate quantity for a long period than a small quantity and even to take a moderate quantity for a long period than for a short, or some factor other than mere duration of feeding has entered by the end of the six month. This result could not have been due to the elimination of the less intelligent animals by the end of 6 months feeding, as only two animals have died by this time and these are too few to have any influence on the group record, even if it were certain that the rats which died were slow rats. Neither could it have been due to the fact that the records of failures are not included in the group average. Adding the records of failures does make the record of the ½ c.c., 6 months group slightly slower than the ½ c.c., 3 months set, but it does little to decrease the disparity between the former and the ¼ c.c., 6 months set, when the record of the single failure belonging to this set is also added to the group average. There are only two possible explanations of the fact that at the end of 6 months feeding ½ c.c. rats are better than ¼ c.c. animals; accommodation to medium doses and not to small doses, and the presence of some undetected disease in

the ¼ c.c. set which did not attack the ½ c.c. animals. As to the first possibility, animals receiving large doses have, to some extent, accommodated to the effects of alcohol feeding but we have no evidence that animals receiving small and medium doses have shown accommodation up to this time. This, however, may be what has occurred in the six months group, as accommodation may come later with a medium than with a large dose. On the other hand the disease factor may have been the cause. Alcoholic animals were shown, when pathological examinations were made, to be very prone to bronchitis. As the severity of the cases was about the same in all alcoholic groups, this has not been cited as a possible cause for behavior differences, the severity of the disease being in no way correlated with the extent of the retardation of the learning process. We have no pathological data concerning this set as examinations were not begun until after the death of all of these rats. An unusually severe case of bronchitis or bronchial pneumonia may have caused, as has been stated, the slowing up of the ¼ c.c. set. We prefer, however, in the light of the facts, to explain the superiority of ¼ c.c. over ½ c.c. rats after 6 months feeding by the hypothesis that after prolonged feeding ¼ c.c. may be as deleterious as ½ c.c. with less possibility of its being accommodated to, or of its actually stimulating to swifter activity. The smaller dose did not stimulate to activity even after 3 months feeding, the larger acted as a stimulant to unusually swift activity in 2 of the 8 cases of animals fed ½ c.c. for 3 months and in 2 of the 4 cases of ½ c.c., 6 months rats.

Our facts, with the few exceptions discussed, are briefly, these:

Alcohol administered to white rats by the feeding method produces a decrease in learning capacity roughly in proportion to the size of the dose administered and the length of the feeding period.

Alcohol decreases learning capacity by (1) decreasing speed of running and (2) increasing the number of learning trials. It does not effect errors per trial.

There are various explanatory conceptions bearing on these results. In the first place, both increase in learning trials and

decrease in speed of running may have been due to the fact that even at the end of 16 days our animals form part of selected groups. In the second place, increase in trials, i.e. poor capacity for elimination, may be due to (A) nervous changes which took the form of lessened retentivity of impressions in the nerve cells themselves, to stronger resistances at the synapses, or even to an overgrowth of sclerotic tissues,* or (B) to the effect of lessened speed. The maze habit is an association of sequent acts. A close temporal contiguity of these acts favors rapidity of association. It is easier to connect two acts occurring immediately together than when they are performed an hour apart. Therefore, speed of running tends to favor the association and slow speed to retard it. Lessened speed may be due to lack of hunger or to the neural changes previously referred to, which might make for insusceptibility to stimuli.

As to the influence of selection, in a previous section we noted that alcohol increases the mortality of animals. As a consequence our groups, especially for the larger doses and longer duration of feeding, are selected animals. For example, the set of 2 ¼ c.c. rats which learned the maze at the end of 3 months feeding, are the surviving members of a group of 43 rats. It is thus entirely possible that the above differential effects between the normal and the various groups of alcoholics are due to this process of selection. Such a result would obtain if it were the intelligent animals that first succumb to the effects of alcohol. As a matter of fact the results are not due to selection. (1) The mortality among ½ c.c. and ¼ c.c. groups is not sufficiently great to account for them. (2) For the 2 c.c. and the 2 ¼ c.c. dosages we determined the correlation between intelligent ability and rate of death for two groups composed of 14 and 16 rats respectively. They were allowed to learn the maze and after records of their relative ability were obtained, alcohol was administered by the usual procedure and the order of their death recorded. The two sets of data were correlated by the ranking method. The correlation data are recorded in Table III.

* No examinations have as yet been made of the nervous systems of alcoholized animals. No discussion of neural changes as a factor in producing lessened speed of acquisition is, therefore, attempted. It is hoped that the necessary data will be in hand shortly.

TABLE III

Correlation between Length of Life and Intelligence

No. of Rats	Dosage	Time	Trials	Errors
7	2 c.c.	+.543	+.426	+.324
7	2 c.c.	+.538	+.652	+.286
8	2 ¼ c.c.	+.191	+.858	+.620
8	2 ¼ c.c.	+.262	—553	+.143

The 2 c.c. animals were members of two groups of 7 each, the 2 ¼ c.c. animals of two groups of 8 each. The record of each group was correlated separately as in the case of both 2 ¼ c.c. and 2 c.c. animals, one group was given one trial a day until the maze was learned, the other two trials a day.

The records show that fairly large positive correlations obtain for the 2 c.c. group between resistance to alcohol and ability to learn the maze, measured in terms of time, trials, and errors. This means that the most intelligent rats were able to live the longest, while the least intelligent animals were the first to succumb. For the 2 ¼ c.c. animals the indices are on the whole lower and one of those for trials is negative. In one group of 2 ¼ c.c. rats the correlations for trials and errors are larger than in either of the 2 c.c. groups, but in the case of trials this is more than compensated for by the negative correlation in the other group, and in the case of errors by the low positive correlation in this second group. While one may doubt a significant positive correlation between resistance and ability to learn in the 2 ¼ c.c. group, yet it is certain that no such inverse correlation obtains as is demanded by the hypothesis that the above differential effects are due to a process of selection. Since it is the least intelligent animals that die first, it is evident that the members of our alcoholic groups that survived the preliminary feeding are the more intelligent animals. That is our alcoholic groups possess more intelligent capacity than the average run of normal animals. The process of selection thus minimizes the deleterious effect of the alcohol, rather than is responsible for it. Such results as we have, therefore, are due to the effect of alcohol on animals to which it was administered and not to the elimination of any particular type of intelligence.

There is only one other factor which remains to be discussed,

some interference with hunger, which was the only motive for running the maze. Alcoholic rats received the same quantity of food as normal animals, and ate with the same speed, indicating that these animals were as hungry as non-alcoholic rats. They did not at any time show the behavior characteristic of animals which are not hungry; that is, they showed no tendency to leave the food dish for at least five minutes after they were placed on the feeding table.

To sum up the material contained in this section: Alcohol has a pronounced effect on the learning process, namely, a decrease in speed and an increase in trials and errors. This effect is not due to the elimination of the more intelligent rats as those eliminated are actually the less intelligent individuals. Neither is this effect due to any interference with the hunger motive. Our only explanation of the differential effects previously discussed is some characteristic change of which we have as yet no anatomical evidence in the structure or function of the nervous system.

IV

The Effect of Parental Alcoholism on the Intelligent Behavior of the Non-Alcoholic Offspring

The second generation of young from alcoholic parents were raised under the same conditions as were the offspring of normal rats. No young of the second, third and fourth generations received alcohol. In both cases every precaution was taken to prevent infection and all animals which showed signs of illness were at once removed from the rest of the group and were not used in experiments. A single exception to this was made in the case of a very few animals which developed bronchitis during the last part of the period in which they were trained in the maze. These were allowed to continue until the maze was mastered as this disease was found not to interfere with the speed of learning where the habit was practically formed. These animals were kept in separate cages, however, in order that the other animals might not be infected.

As a matter of convenience all second and third generation young from alcoholic parents will hereafter be termed "non-alcoholic" young, and all young from normal stock, "normals."

The effect of parental alcoholism on the intelligent behavior of the surviving offspring is shown in Table III. At the age of 66 days all second generation animals were trained in the maze used in Section I. They were given one trial per day until four out of five consecutive runs had been made without error. Time, trials, and errors before the maze was mastered were the criteria used in judging relative intelligence.

The records of the second generation bring out clearly the fact that parental alcoholism does affect the intelligent behavior of non-alcoholic offspring, even when it is confined to the male parent alone. In Sets I, II, V and VI the effect of parental alcoholism is uniformly bad. Set I, the offspring of a 2 c.c. male under the influence of alcohol for 5 months and a subsequent

TABLE IV

The Effect of Parental Alcoholism on the Intelligent Behavior of the Second Generation

Set	Male parent	Female parent	No. failing	No. learning	Total trials	M.V.	Total errors	M.V.	Total time	M.V.	Ave. time per trial	Ave. error per trial
I.	2 c.c. 7 mos.	Normal	0	4	35.2	10.8	240.0	62.4	37281.5″	51751.8″	1059.0″	6.8
II.	½ c.c.	5 ½ mos.	0	4	23.5	9.5	162.2	69.3	8754.7″	6621.3″	372.5″	6.8
III.	¼ c.c.	39 days	0	4	8.0	1.0	70.5	13.0	1417.5″	574.0″	177.1″	8.8
IV.	¼ c.c.	4 mos.	0	5	9.5	3.3	28.2	8.5	300.0″	98.4″	31.7″	3.+
V.	¼ c.c.	6 mos.	0	3	28.0	1.+	153.6	32.8	24001.6″	11251.1″	857.2″	5.4
VI.	¼ c.c. 6 mos.	2 normals	0	9	16.7	4.1	119.1	35.0	9767.6″	1652.0″	584.8″	7.1
VII.	½ c.c. 4 mos.	Normal	0	6	6.6	1.6	28.5	7.0	694.0″	226.2″	105.1″	4.3
VIII.	Normal	Normal	0	15	8.0	2.5	44.5	17.1	1237.2″	1156.7″	154.6″	5.6

non-alcoholic period of 2 months and a normal female whose offspring, when mated with a normal male showed no abnormalities, take 27.2 more trials, and 35944.3″ more time, and make 195.5 more errors than young from the normal matings. Set II, the result of a mating between two ½ c.c. animals under the influence of alcohol for 5 months, take 15.5 more trials and 7517.5″ more time, and make 117.7 more errors than do normals. Set V, the offspring of ¼ c.c. 6 months rats, take 22764.4″ more time, 20 more trials and make 109.1 more errors than do normals.

On Set III parental alcoholism has little or no definite effect. The animals are slightly inferior to normals in time and number of errors but take the same number of trials to master the maze. Such differences as are present are such as may be accounted for on the basis of individual variations between groups.

An interesting feature of these results is the behavior of Set IV, the offspring of rats fed ¼ c.c. for 4 months before mating, and of Set VII, the offspring of a ½ c.c. 4 months male and a normal female. These animals take much less time to master the maze and make much fewer errors than do normals of the same generation. Set IV is slightly inferior to normals in the matter of trials, but Set VII is markedly superior in all three measures. This does not mean that all animals in Set IV were superior to normals. Some, as indicated by the mean variation, were unusually slow. The mean variation here is not large because the group is large since this group is actually smaller than the normal group and its mean variation is much larger. Neither is the superiority on the average of either group due to the fact that the groups are small, since compared with the third generation group of the same age, which is even smaller, they are still superior. We have, then, two phenomena, neither of which seems to be due to the relative size of the groups, which require an explanation; (1) the large mean variation, especially in time, in the records of Set IV and, (2) the fact that animals from alcoholic stock were actually better than normal animals raised under the same conditions. As to the former of these two facts there is little to state. This phenomenon is probably due to the difference in germ cells which enables some to resist entirely the

poisonous effect of the alcohol and others to feel its effect only slightly. In the former case young produced from these cells would not be below normal, in the latter the young produced would show certain characteristic behavior changes. Hence there should be present in the surviving young all degrees of defect. This would explain the large mean variation in the time taken by Set IV to master the maze.

The increased speed of learning indicated by the group averages of Sets IV and VII may have been due (1) to a heightened nervous excitability analogous to that found by Lashley[5] in strychinized animals; (2) to the elimination of less strong germ cells and a consequent bettering of the stock, as Dr. Pearl suggests was the case with alcoholized fowl; (3) to some beneficial effect of the alcohol on the parent animals.

The third of these possibilities can be dismissed without a lengthy discussion. The results previously cited of examinations of the generative organs of alcoholized animals, together with the fact that not even those receiving the minimum dose lived longer than one year, would banish at once any hypothesis which took into account the possible beneficial effects of alcohol; while at the same time they bring much to the support of the first hypothesis, namely, that alcoholization may have eliminated the less strong germ cells. In no case of a ¼ c.c. or ½ c.c. per day animal were all spermatozoa normal, and in many cases the seminiferous tubules were packed with spermatids which degenerated without attaining maturity. Obviously numerous germ cells were eliminated by reason of alcoholism. This alone would be sufficient to explain our results, but it is perfectly possible that the nervous excitability which Lashley found in strychinized animals was also a factor with offspring of alcoholics since all these animals which learned the maze in less time trials than the average normal were unusually active and moved with great rapidity.

An examination of the heart, lungs, kidney and adrenals, stomach and liver showed no disease present which would in any way account for behavior differences—these must, therefore, be due to parental alcoholism alone.

To summarize the material contained in this section:

Parental alcoholism may have as its effect an increase in average time and number of trials necessary to master the maze. This occurred in Sets I, II, V, and VI.

Parental alcoholism may have a beneficial effect on speed of learning as measured by a decrease in time taken and errors made before the maze is mastered; this occurred in Sets IV and VII; or it may produce no definite effect whatsoever, as was the case with Set III.

All inferior groups were from parent stock fed alcohol for from 5 to 7 months; both superior groups and the single set on which parental alcoholism had no effect were from parent stock fed for four months or less.

Though there is not very much evidence as to the effect of large as compared with small dosages, such evidence as we have points to the conclusion that the effect of parental alcoholism is partially dependent on the size of the dose administered to parent animals.

Long feeding and medium or large doses act detrimentally; short feeding periods and small doses have either no effect or a beneficial one.

Examinations of the generative tracts of all animals except those in Set I and the males in Set II, all of which died before examinations could be made, demonstrated no definite changes. As might have been expected, a normal number of young were produced.

V

The Effect of Parental Alcoholism on the Intelligent Behavior of the Third and Fourth Generation

Third generation rats were trained in the same maze and with the same procedure as were the parent animals.

The behavior of the third generation resembles closely that of their parents (Table V). Here, as in the parent animals, relative intelligence is dependent upon the degree of alcoholism. Set II, 2nd generation, is less intelligent than Sets III and IV, 2nd, and has less intelligent offspring than either of these sets. In all three cases the offspring are less intelligent than the parent animals.

The offspring of Set II are much less intelligent than the parent animals. The fact that they take fewer trials and make fewer errors than do the parent animals, is more than compensated for by the number of failures. Here, as in the first generation, the total time, trials and errors would be greatly increased were the records of failures added to the group average. As compared with normal third generation animals, their set takes 9.4 more trials and 19014.2″ more time and makes 92.0 more errors even with the records of failures not included in the group average. In number of failures the third generation bears closer resemblance to the first than to the second generation. The second contains no failures whereas the first contains 2 and the third 4. This is the only respect in which the behavior of the third generation bears any resemblance to the behavior of the first generation of the same stock. In all respects the third generation of this stock is approximately as inferior to the second generation as the second was to the first. That is to say, when animals receiving ½ c.c. for five months are inbred the defectiveness present in the first generation is somewhat increased in the second and when this generation is, in turn, inbred the defectiveness is even more increased in the third. When the females of

Set II, 2nd, were crossed with a normal male, the third generation of offspring were, as contrasted with those resulting from inbreeding, slightly superior to normal third generation animals in trials and total time and only slightly inferior in trials. Crossing this alcoholic stock with normal, therefore, tends to eliminate the defectiveness.

The offspring of Set III, 2nd, a set which had showed slightly, if at all, the effect of parental alcoholism, are somewhat inferior to the normal third generation animals. A male of this set crossed with a normal female produced young superior to normal in all respects.

The offspring of Set IV, 2nd, a set which was itself superior to the normal second generation animals, are inferior to normal third generation animals and therefore inferior to the parent animals in total time taken to master the maze. In total errors this set is inferior to the parent animals but still superior to normal animals, in trials it is superior both to the parent animals and to normals. It is therefore slightly superior to the parent set in one measure but inferior in two. Inbreeding has produced an increased defectiveness. A male of this group mated with a normal female produced young even more inferior to the normal except in the matter of time. In this respect mating with a normal has been advantageous. As has frequently been stated increase in total time is, in all generations, the most characteristic effect of alcohol feeding. As animals of all generations ate as much as normals and remained as long by the food receptacle, there was no tendency to slow up because of lack of hunger.

To sum up: All alcoholic strains show an increased defect in the third generation when both parents are alcoholic. In two of the three cases the defect is less marked in the second generation than in the third.

The offsprings of matings between alcoholic and normals are superior to the alcoholic parent and in one of three cases slightly superior to normals as well.

In general, in the third generation as in the first and second generations, the most characteristic effect of the alcohol is to

TABLE V

The Effect of Alcoholizing the First Generation on the Intelligent Behavior of the Third

First Generation											
Parent of male parent	Parent of female parent	No. of failures	No. learning	Total trials	M.V.	Total errors	M.V.	Total time	M.V.	Ave. time	Ave. errors
¼ c.c. 39 days	¼ c.c. 39 days	0	11	8.3	3.4	55.8	29.4	4516.8″	5232.4″	544.1″	6.7
¼ c.c. 4 mos.	¼ c.c. 4 mos.	0	11	6.0	2.9	34.7	14.1	5705.2″	7313.9″	911.5″	5.7
½ c.c. 5 mos.	½ c.c. 5 mos.	4	5	16.4	7.6	148.0	46.4	20357.4″	9481.6″	124.0″	9.0
¼ c.c. 39 days	Normal	0	3	4.6	1.1	34.6	5.8	397.3″	94.4″	86.7″	7.5
¼ c.c. 4 mos.	Normal	0	6	9.8	1.7	54.5	10.6	3838.0″	2972.0″	391.6″	5.5
Normal	½ c.c. 5 mos.	0	10	9.9	2.3	54.0	11.4	1152.8″	356.1″	116.0″	5.4
Normal		0	5	7.0	2.4	56.0	7.8	1343.2″	872.1″	191.8″	8.0

lessen the speed with which the maze is run. That is to say, alcoholic animals and their non-alcoholic offspring tend to stay much longer in the maze than do normal animals of the same age raised under the same conditions and run at the same time. A marked increase in the number of trials taken to master the maze and the number of errors made occurs only where the feeding period of the parent animals has been of long duration but in all sets of first, second, and third generation rats; with three exceptions, Sets IV and VII of the second generation and Set IV of the third generation; alcoholic animals take more time in the maze than do normal rats of the same generation. In every case the normal animals were raised under the same conditions and were of exactly the same age.

Had the deleterious effect of the alcohol stopped with the second generation, malnutrition or disease, without any definite germ cell modification might have been assigned as the cause. This was not the case. Set one of the second generation was sterile. Set II produced young much less intelligent than its parents when inbred and less intelligent than normals in one measure when crossed with normal males. Sets III and IV produced young whose behavior somewhat resembles their own even when mated with normal females.

Obviously the behavior differences present in the first and second generation are present also in the third.

The records of the fourth generation show but slight tendency to reproduce in the offspring the type of behavior which characterized the parent animal. Only two sets of the fourth generation animals have been obtained. Set 1, fourth generation, is the offspring of the single fertile male of Set III, third generation, and a female of the same set. Set II is the offspring of a male and female of the third generation, Set II.

As there were no normal fourth generation offspring[1] the behavior of abnormal Sets I and II can only be compared with the behavior of the normal third generation set.

There is still a tendency to slower adaptations than those present in normal animals in Set I, whose parents were much slower than normals. Trials taken and the errors made are both slight-

TABLE VI.

The Effect of Alcoholizing the First Generation on the Intelligent Behavior of the Fourth

Set	No. of failures	No. learning	Total trials	M.V.	Total errors	M.V.	Total time	M.V.	Ave. time	Ave. errors
I	0	7	9.0	4.6	62.8	21.4	1433″	898″	170.3″	8.9
II	0	6	8.5	2.2	44.8	12.5	1848″	931″	212.+	5.2
Normal	0	5	7.0	2.4	56.0	7.8	1343″	827.1″	191.8	8.0

[1] The normal third generation set died of a contagious disease which killed, also, several alcoholic sets.

ly greater than those of the second and third generation normals, but such a small variation from normal as we have in this set may be due to individual differences rather than to alcoholism. Set II, the offspring of Set II of the third generation, which showed itself superior to normals, is still superior in one measure, errors made, and but slightly inferior in number of trials and in length of time taken before the maze was learned. On the whole it seems better to assume that such defects as were present in the second and third generation have been bred out in the fourth.

That the defects present in the stock producing Set I, 4th, have been bred out through the non-fertility of a large proportion of the stock is obvious from a study of the proportion of fertility present in the third generation of these animals. As has been stated in Section I, the section containing pathological data, only one male and one female of this set produced young though attempts were made to breed the remaining animals both with rats of the same set and with normals. It is also probable that the defect has been bred out in Set II of the fourth generation though the data in this case is, because of the short period during which these animals were mated, far from conclusive.

To summarize the material contained in Sections III, IV, and V:

The defects present in the first generation are, in general, transmitted to the second and third generation.

All doses administered for long periods have a markedly deleterious effect on the offspring. This effect is present to some degree in the second generation and to an even greater degree in the third. It has bred out by the fourth generation.

Small doses administered for short periods have either no effect on the progeny or are actually beneficial.

Some stocks have bred out through total sterility. No 2¼ c.c. animals produced offspring. These were, therefore, eliminated in the first generation. Only one 2 c.c. animal, a male, had young which survived and these were by a normal female. All of the four surviving offspring were sterile. This stock was therefore eliminated in the second generation. The three surviving young of ¼ c.c., 6 months parents, were also sterile and were eliminated in the second generation.

Sets VI and VII, 2nd generation, and Sets IV, V, and VI, 3rd generation, were born too late in the last year during which this experiment was in progress to make any extensive tests of fertility possible.

In some cases the defect in the stock has been bred out through partial sterility. This has probably occurred in the case of Sets IV and VII, 2nd generation and Sets I and II, 4th generation. Set IV were born of a mating between two ¼ c.c., 4 months animals, and Set VII of a mating between a normal female and a ½ c.c., 4 months male, Set I, 4th generation, came from ½ c.c., 5 ½ months stock and Set II of ¼ c.c., 39 days stock. We have only anatomical evidence to cite in the cases of Sets IV and VII, 2nd generation. Examinations of the generative tracts of alcoholized animals showed, as has been stated, that in no instance were all the spermatozoa of ¼ c.c. or ½ c.c. rats normal, and in many instances the seminiferous tubules were packed with spermatids which degenerated without attaining maturity. Many germ cells were, therefore, eliminated as a result of alcoholism.

As to Set II, 4th generation, the evidence is not conclusive as no extensive tests of fertility were made. It is possible, however, that the improvement in this set may again be due to the failure to function of the less strong germ cells. In the case of Set I, 4th generation, we have a clear evidence that the defect in this stock has bred out through partial sterility. Only one female and one male of this set had young, though attempts were made to inbreed and to cross with normal males and females. Three males and three females of Set I, 3rd generation, were left

in the same cage for 6 months; 2 males were left in the same cage with 2 normal females and 2 females of this set were left in the same cage with 2 normal males for three months. No young resulted, though the animals were left together at the mating season. Set I, 4th generation, was therefore the result of some selective process.

It is hardly necessary to point out further the resemblance between animals of the 1st, 2nd, 3rd and 4th generations of the same alcoholized stock, both as to intelligent behavior and as to relative fertility.

Taking into account the results of the examinations of the testicles of alcoholized rats cited in the article previously mentioned i.e.—that headless and tail-less spermatozoa are present in large numbers in the tubules and that spermatids are found in a state of complete arrest and sometimes degeneration, all of which is sufficient evidence that the germ cell itself is affected by alcoholization; and the behavior of our animals, both as to reproductive capacity and intelligent adaptation, the author feels that the results warrant the statement that alcohol can affect a thoroughly healthy stock in such a manner as to produce characteristic changes in the offspring, both as to intelligent behavior and as to degree of fertility. These changes are present, not only in the second generation but also in the third and to some extent in the fourth generations, and are apparently mediated by germinal changes.

VI

Conclusions

1. Alcohol has a marked effect on the general bodily health of white rats, causing a very slow gain in weight as compared with normals and in some cases an actual loss though the animals were in the growing period.

2. This retardation in growth is inherited by the non-alcoholic offspring of alcoholic animals.

3. Alcohol also has a pronounced effect on length of life. Large doses caused the death of from 74 per cent to 82 per cent of the animals and smaller doses of from 10 per cent to 24 per cent.

4. Large doses of alcohol cause complete sterility, small doses cause a decrease in the number of litters as compared with normals, and of the number of viable young in these litters. Examination of the generative tract of alcoholics gives a clue to the cause of these abnormalities.

5. Alcohol administered for 16 days and 3 months has a deleterious effect upon the speed of running and rate of learning the maze. The effect is roughly in proportion to the size of the dose administered and the duration of the feeding period.

6. Small doses, ½ c.c. and ¼ c.c. per day per rat, fed for 6 months do not effect behavior in proportion to the size of the dose. The smaller dose has a more deleterious effect than the larger. This is probably due to a tendency to accommodate more rapidly to the larger dose.

7. Parental alcoholism results in lessened speed of running and rate of learning the maze when the parental alcoholism has continued for a prolonged period, and in increased speed of running and rate of learning when the dose administered to parent animals has been small and the feeding period short.

8. The effects of parental alcoholism present in the second generation are transmitted to the two succeeding generations, but defects present in the preceding generations tend to breed out by the fourth generation.

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